Journal of Consulting and Clinical Psychology
2006, Vol. 74, No. 4, 707–713

DOI: 10.1037/0022-006X.74.4.707

Longitudinal Analysis of the Relationship Between Symptoms and Quality of Life in Veterans Treated for Posttraumatic Stress Disorder

Paula P. Schnurr
National Center for Post-Traumatic Stress Disorder, U.S. Department of Veterans Affairs, and Dartmouth Medical School

Andrew F. Hayes
The Ohio State University

Carole A. Lunney
National Center for Post-Traumatic Stress Disorder, U.S. Department of Veterans Affairs

Miles McFall
Veterans Affairs Puget Sound Healthcare System and University of Washington School of Medicine

Madeline Uddo
New Orleans Veterans Affairs Medical Center

This study examined how change in posttraumatic stress disorder (PTSD) symptoms relates to change in quality of life. The sample consisted of 325 male Vietnam veterans with chronic PTSD who participated in a randomized trial of group psychotherapy. Latent growth modeling was used to test for synchronous effects of PTSD symptom change on psychosocial and physical health-related quality of life within the same time period and lagged effects of initial PTSD symptom change on later change in quality of life. PTSD symptoms were associated with reduced quality of life before treatment. There were synchronous effects of symptom change on change in quality of life but no significant lagged effects. Results indicate the importance of measuring quality of life in future investigations of PTSD treatment.

Keywords: posttraumatic stress disorder, quality of life, military veterans, group psychotherapy

Individuals with posttraumatic stress disorder (PTSD) experience reduced quality of life (e.g., Magruder et al., 2004; Schonfeld et al., 1997; Stein, Walker, Hazen, & Forde, 1997). For example, a recent study found that 59% of PTSD patients had severe quality of life impairment, which was comparable to 63% of patients with major depression (Rapaport, Clary, Fayyad, & Endicott, 2005). Furthermore, prospective cohort studies have found that initial PTSD predicts poor life quality at subsequent follow-up intervals (Holbrook, Hoyt, Stein, & Sieber, 2001; Michaels et al., 1999; Zatzick, Jurkovich, Gentilello, Wisner, & Rivara, 2002). There also is growing evidence that quality of life improves following treatment for PTSD (e.g., Foa et al., 1999; Rapaport, Endicott, & Clary, 2002; Tucker et al., 2001).

Including quality of life as an outcome in studies of PTSD symptom-focused treatment reflects the assumption that reduced quality of life is secondary to symptoms. However, the observation of improvements in both symptoms and quality of life is only an indirect reflection of the relationship between these domains. Furthermore, such studies fail to capture lagged relationships. Time may be needed in order for immediate improvement in symptoms to affect quality of life. For example, a person whose avoidance and irritability decrease following treatment may not show improvements in social functioning for months afterward because of the time it takes to make and rebuild friendships. The effects of PTSD symptom improvement on physical health might be delayed given the complexity of factors hypothesized to underlie the relationship between PTSD and poor physical health (Schnurr & Green, 2004).

We used latent growth modeling to fit a longitudinal model to examine how change in PTSD symptoms relates to change in quality of life. Data came from veterans with chronic PTSD who took part in a randomized clinical trial of group psychotherapy that compared trauma-focused and present-centered approaches (Schnurr, Friedman, Lavori, & Hsieh, 2001). The year-long treatment consisted of 30 weekly sessions to help patients reduce symptoms, followed by 5 monthly sessions to help patients maintain their gains. Both conditions showed significant and compara-

Paula P. Schnurr, National Center for Post-Traumatic Stress Disorder, U.S. Department of Veterans Affairs, and Department of Psychiatry, Dartmouth Medical School; Andrew F. Hayes, School of Communication, The Ohio State University; Carole A. Lunney, National Center for Post-Traumatic Stress Disorder, Department of Veterans Affairs; Miles McFall, VA Puget Sound Healthcare System and Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine; and Madeline Uddo, New Orleans VA Medical Center.

This research was supported by a grant to Paula P. Schnurr from the U.S. Veterans Affairs Cooperative Studies Program. A version of the paper was presented in November 2005 as a poster at the annual meeting of the International Society for Traumatic Stress Studies, Toronto, Canada. The views expressed in this article are those of the authors and do not necessarily represent the views of the National Center for Post-Traumatic Stress Disorder or the U.S. Department of Veterans Affairs.

Correspondence concerning this article should be addressed to Paula P. Schnurr, National Center for Post-Traumatic Stress Disorder (116D), U.S. Veterans Affairs Medical Center, White River Junction, VT 05009. E-mail: paula.schnurr@dartmouth.edu

ble improvement in PTSD symptoms. Although treatment was not associated with improved quality of life, there were marked intra-individual changes on all of the measures used.

Figure 1 depicts our model, which is described in greater detail below. The two treatment phases in the original study enabled us to examine whether there were (a) synchronous effects of PTSD symptom change on quality of life change within the same phase and (b) lagged effects of PTSD symptom change during initial weekly treatment on change in quality of life during subsequent monthly treatment. We expected that higher PTSD severity would be associated with poorer quality of life at baseline and that there would be synchronous change during both phases. Prior evidence did not provide a basis for hypothesizing that there also would be lagged effects, although such effects are logically plausible. Quality of life includes psychosocial and physical elements (Gladis, Gosch, Dishuk, & Crits-Christoph, 1999; Mendlowicz & Stein, 2000). We fitted a separate model for each domain because it was possible that relationships between changes in symptoms and in quality of life would differ across domains.

Method

Participants

Participants were 325 male Vietnam veterans with PTSD in a randomized clinical trial of group therapy for PTSD (Schnurr et al., 2001, 2003). They had to agree to terminate other PTSD treatment except for 12-step programs and, if on psychoactive medication, be on a stable regimen for 2 months before the study. Exclusion criteria, based on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*]; American Psychiatric Association, 1994) were the following: current or lifetime psychotic disorder, mania, or bipolar disorder; current major depression with psychotic features; current alcohol or drug dependence; unwillingness to refrain from substance abuse at treatment or work; significant cognitive impairment; and severe cardiovascular disorder.

Thirty-five veterans from the original sample of 360 were excluded because they did not participate in any follow-up assessments. These 35 men were comparable to the 325 included men on almost all measures, including the primary PTSD outcome (Schnurr et al., 2003).

Participants' average age was 50.69 years ($SD = 3.68$). Two hundred fifteen (66.2%) were White, 74 (22.8%) were Black, and 36 (11.0%) were from other ethnic groups; 23 across all groups (7.1%) were Hispanic. Most had graduated from high school (90.2%, $n = 290$), and half (50.2%, $n = 163$) were unemployed. The majority (70.8%, $n = 230$) received Veterans Affairs (VA) disability payments for a physical or mental problem; 60.6% ($n = 197$) were service-connected for PTSD.

Measures

PTSD symptoms. Three indicators were used to assess PTSD symptoms. The Clinician-Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001) is a structured interview in which the frequency and intensity of each of the 17 *DSM-IV* PTSD symptoms is rated on a 5-point scale (ranging from 0–4). Scores are summed to create a total severity measure. A change of ≥10 points (roughly 0.5 *SD*) is defined as a clinically significant change. The PTSD Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) questionnaire contains the 17 *DSM-IV* PTSD symptoms, rated on a 5-point scale ranging from 1 (*not at all*) to 5 (*extremely*). It has very good sensitivity and specificity for a diagnosis of PTSD. The 12-item General Health Questionnaire (GHQ; Goldberg, 1992) has good sensitivity and specificity as a measure of PTSD in a traumatized sample (McFarlane, 1986). Symptoms are rated on a 4-point scale ranging from 1 (*much less than usual*) to 4 (*much more than usual*). Higher scores reflect higher symptom severity for each of these scales.

Quality of life. Quality of life was measured with the Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992) and the Quality of Life Inventory (QOLI; Frisch, 1994). Both instruments have excellent psychometric properties (Frisch, 1994; Frisch et al., 2005; Gladis et al., 1999;

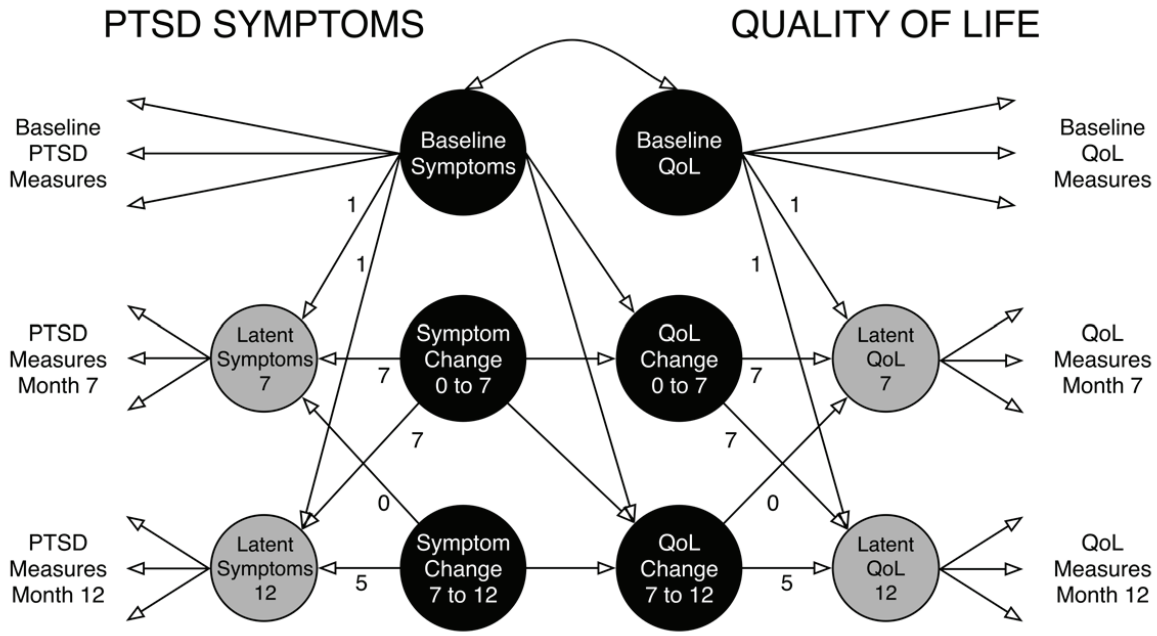

Figure 1. Multivariate latent growth model with cross-domain estimation of change parameters. PTSD = posttraumatic stress disorder; QoL = quality of life.

McHorney, Ware, Rogers, Raczek, & Lu, 1992). The SF-36 has been recommended as the best all-purpose instrument for assessing quality of life in those with anxiety disorders (Mendlowicz & Stein, 2000). SF-36 scores range from 0 to 100, with higher scores reflecting better outcomes. Four of its eight scales reflect mental/emotional/social functioning and well-being; the other four scales reflect physical functioning and health perceptions. On the QOLI, each of 16 aspects of life (e.g., work, friendship) is rated in terms of satisfaction (−3 = *very dissatisfied* to 3 = *very satisfied*) and importance (0 = *not at all important* to 2 = *extremely important*). For items with non-zero importance ratings, each satisfaction rating is weighted by its importance value and then averaged to form a composite that ranges from −6 (most negative) to 6 (most positive).

As indicated above, our analyses separately examined psychosocial and physical domains. There were three indicators of psychosocial quality of life: the QOLI and the SF-36 Social Functioning and Role—Emotional scales. We selected these SF-36 scales because their content explicitly addresses psychosocial functioning; Mental Health and Fatigue were excluded to avoid the observation of spurious relationships with the PTSD measures. The 4 physical health scales from the SF-36 were used as indicators of physical health-related quality of life: Physical Functioning, Role—Physical, Bodily Pain, and General Health.

Procedure

Written informed consent was obtained from each participant after the study procedures had been explained, prior to data collection and the initiation of treatment. Across 10 VA Medical Centers, participants were randomly assigned to either trauma-focused group therapy (TFGT; Foy, Glynn, Ruzek, Riney, & Gusman, 1997) or present-centered group therapy (PCGT). In TFGT, exposure is conducted in a group context that includes psychoeducation, cognitive restructuring, relapse prevention, and coping skills training. PCGT includes nonspecific and supportive kinds of interventions in order to control for the nonspecific benefits of the group experience. It does not include exposure or the other elements of TFGT.

Each treatment group consisted of 6 participants. Two master's- or doctoral-level clinicians conducted treatment using a manualized protocol. Weekly group therapy was provided for 30 weeks, followed by monthly group booster sessions for 5 months. Assessments were performed at baseline, 7 months (the end of weekly sessions), and 12 months (the end of monthly sessions). Assessments were done by a master's- or doctoral-level clinician blind to group assignment.

Statistical Analysis

We estimated separate structural equation models for the two quality of life domains, each with two components: (a) a multivariate latent growth measurement model estimating baseline and change in PTSD symptoms and quality of life during the study and (b) a structural component linking the baseline and change estimates from the latent growth models (e.g., MacCallum, Kim, Malarkey, & Kiecolt-Glaser, 1997; Willet & Sayer, 1994). The model is shown in Figure 1.[1] We used a two-step approach, first estimating a good-fitting measurement model, then estimating the structural component. Parameter estimates were derived through maximum likelihood using M-plus (Muthén & Muthén, 1998).[2]

The measurement component of the model was estimated separately for PTSD symptoms and for the two quality of life domains to estimate change in PTSD symptoms and quality of life over time, with change treated as a random effect. Underneath the latent growth model is a factor analytic model of PTSD symptoms and quality of life, with each construct measured with the same indicators measured over time (three for PTSD and psychosocial quality of life; four for physical health-related quality of life). Each participant's change estimates represented his average monthly change in latent symptoms or quality of life (a) between baseline and 7 months and (b) between 7 and 12 months.[3] Changes over time are indexed relative to the baseline factor mean (constrained to zero). Positive values indicate an increase and negative values indicate a decrease in symptoms or quality of life.

The structural model was used to examine (a) synchronous effects of PTSD on quality of life, that is, whether change in PTSD symptoms predicted change in psychosocial or physical health-related quality of life during the same treatment phase and (b) lagged effects, that is, whether change in symptoms during initial weekly treatment predicted change in quality of life during subsequent monthly treatment.[4] We also assessed whether change in quality of life depended on baseline symptoms. The structural parameters were estimated as fixed effects.

In exploratory analyses, we tested the possibility of causal paths from change in quality of life to symptom change by reversing the direction of the paths in the structural model. That is, PTSD symptoms were treated as the outcome, and paths went from baseline quality of life and the two quality of life change estimates to the two PTSD symptom change estimates. These analyses were the same as the primary analyses in all other respects.

Results

Information about the latent growth measurement model is presented in Table 1. All three measurement models fit well according to conventional cutoff criteria (Hu & Bentler, 1999). Except for baseline psychosocial quality of life, reliabilities reached traditionally acceptable levels, although reliability was slightly lower for the psychosocial domain than for the physical domain.

The observed means and factor loadings at each time period are presented in Table 2. The observed means show that the sample had significant PTSD and poor psychosocial and health-related quality of life according to definitions or norms for the individual measures. For example, on the CAPS, scores of 40–59 are considered "PTSD" and scores of 60–79 are considered "severe PTSD" (Weathers et al., 2001). On the SF-36, scores for the current sample fell between 1 and 2 *SD*s below norms for the U.S. general population, which range from 70.1 to 83.0 on the four

[1] Not depicted in Figure 1 are the error correlations between each indicator over time to account for associations between the same indicator not attributable to change in the latent variable over time. Model fit was always improved by freely estimating these covariances rather than fixing them to zero. Residuals in the estimation of the quality of life change parameters are also not shown in Figure 1 but are represented as d1 and d2 in Figure 2.

[2] Full information maximum likelihood (FIML) was used to handle missing data, which was assumed to be missing at random. When listwise deletion was used, there were no qualitative changes to the results.

[3] To interpret these estimates as desired and to identify the model, several constraints had to be imposed: (a) factor loadings for each indicator were constrained to be constant over time, making the scores on the latent variables comparable over time; (b) intercepts of the model estimating each indicator were constrained to be constant over time; (c) factor intercepts were fixed to zero; (d) the factor loading for the first indicator was fixed to 1 at each time point; (e) factor variances were fixed at 1; and (f) the baseline factor mean was constrained to zero.

[4] We fit an additional structural model that included age, therapy type, ethnicity (White vs. non-White) and education as additional predictors of change in quality of life. Because none of these variables significantly predicted quality of life change and none of the structural coefficients linking change to change were qualitatively affected by including these predictors, we do not discuss this more complicated model.

Table 1
Latent Growth Measurement Model Results

Variable	PTSD symptoms	Psychosocial quality of life	Physical health-related quality of life
Latent variable reliabilities			
Baseline	0.76	0.65	0.81
7 months	0.87	0.72	0.82
12 months	0.90	0.70	0.84
Latent means			
Baseline	0.00	0.00	0.00
7 months	−4.45	0.61	−0.99
12 months	−5.55	1.01	−2.36
Mean latent monthly change			
Baseline to 7 months	−0.64***	0.09	−0.14
7 to 12 months	−0.22	0.08	−0.28
Variances			
Baseline	204.83***	398.95***	414.95***
Baseline to 7 months	4.19***	4.70***	2.22***
7 to 12 months	6.22***	5.97***	4.30***
Goodness of fit			
CFI	0.99	1.00	1.00
RMSEA	0.05	0.01	0.03
χ^2	39.03*	23.92	64.22
df	23	23	51

Note. $N = 325$. PTSD = posttraumatic stress disorder; QoL = quality of life; *CFI* = comparative fit index; *RMSEA* = root mean square error of approximation. Reliability for each construct was estimated as $(\Sigma_j\lambda_j)^2/[(\Sigma_j \lambda_j)^2+\Sigma_j(1 - \lambda_j^2)]$, where λ_j is the standardized factor loading for indicator j.
* $p < .05$. *** $p < .001$.

physical health subscales and from 83.1 to 83.6 on the two psychosocial health subscales we used (QualityMetric Incorporated, 2006).

The coefficients for mean latent monthly change indicate a statistically significant average reduction in PTSD from baseline to the end of weekly treatment but no significant change during monthly treatment (Table 1). The intraindividual change was substantial, for example, on the CAPS, 38.2% of the sample had clinically significant improvement and 18.6% had clinically significant worsening from 0 to 7 months. There was no average change in either quality of life domain during either phase; however, the statistically significant variances reflect meaningful individual differences comparable to those observed on the CAPS. For example, on the QOLI, 24.5% of the sample improved and 20.7% worsened from 0 to 7 months by ≥0.50 *SD*. During the same time, 16.5% to 25.8% improved and 19.6%–26.4% worsened by ≥0.50 *SD* on the SF-36 subscales.

In Figure 2 we graphically display the structural model and structural parameter estimates for psychosocial and physical quality of life domains. Although the figure displays only the structural model, it is important to keep in mind that the structural model and the measurement model are combined at this stage of the analysis into a single omnibus model. The overall models fit the data well by traditionally accepted standards, *CFI* = .97, *RMSEA* = 0.06, $\chi^2(121) = 248.16$, $p < .001$, for psychosocial quality of life, and *CFI* = .98, *RMSEA* = 0.04, $\chi^2(176) = 272.42$, $p < .001$, for physical health-related quality of life.

At baseline, higher PTSD symptom severity was associated with poorer psychosocial quality of life (Figure 2). There were synchronous effects of PTSD symptom change from 0 to 7 months and from 7 to 12 months. Improvement in symptoms was associated with an improvement in quality of life. The lagged effect of PTSD symptom change was not statistically significant, that is, symptom change from 0 to 7 months did not predict quality of life change from 7 to 12 months. Baseline PTSD symptoms significantly predicted change in psychosocial quality of life from 0 to 7 months, but not from 7 to 12 months. Participants with higher PTSD symptom severity at baseline improved in psychosocial quality of life during weekly treatment more so than those with relatively fewer PTSD symptoms. Overall, the model explained 77% and 49% of the variance in change in psychosocial quality of life from 0 to 7 months and from 7 to 12 months, respectively.

The results for physical health–related quality of life were highly similar to the results for psychosocial quality of life (Figure

Table 2
Latent Variable Indicator Means, Standard Deviations, and Factor Loadings

Variable	Observed means			Factor loadings		
	Baseline	7 months	12 months	Baseline	7 months	12 months
PTSD symptoms						
CAPS	81.22 (18.42)	75.15 (22.42)	74.40 (24.31)	.76 (1.00)	.84 (1.00)	.86 (1.00)
GHQ	33.07 (6.92)	31.39 (7.28)	31.56 (7.50)	.60 (0.29)	.73 (0.29)	.78 (0.29)
PCL	62.22 (11.74)	60.36 (13.41)	59.55 (13.85)	.79 (0.63)	.91 (0.63)	.94 (0.63)
Psychosocial quality of life						
Social functioning	46.05 (27.74)	46.00 (26.39)	47.76 (27.51)	.73 (1.00)	.80 (1.00)	.77 (1.00)
Role—emotional	22.19 (33.22)	22.57 (34.83)	20.94 (33.55)	.59 (0.99)	.61 (0.99)	.61 (0.99)
QOLI	−0.18 (2.07)	−0.06 (1.93)	−0.12 (1.93)	.54 (0.06)	.61 (0.06)	.60 (0.06)
Physical quality of life						
Physical functioning	62.88 (27.99)	61.38 (28.05)	60.08 (27.74)	.75 (1.00)	.75 (1.00)	.76 (1.00)
Role—physical	36.88 (41.98)	34.47 (40.00)	33.82 (40.89)	.70 (1.44)	.73 (1.44)	.74 (1.44)
Bodily pain	43.59 (25.96)	43.41 (27.12)	41.93 (24.78)	.72 (0.93)	.72 (0.93)	.77 (0.93)
General health	42.68 (23.41)	42.17 (23.62)	40.63 (24.16)	.71 (0.82)	.71 (0.82)	.72 (0.82)

Note. PTSD = posttraumatic stress disorder; CAPS = Clinician-Administered PTSD Scale. GHQ = General Health Questionnaire; PCL = PTSD Checklist; QOLI = Quality of Life Inventory. Numbers in parentheses beside observed means are standard deviations. Numbers in parentheses beside factor loadings are unstandardized factor loadings. Factor loadings are from the measurement model constraining factor loadings to be invariant over time.

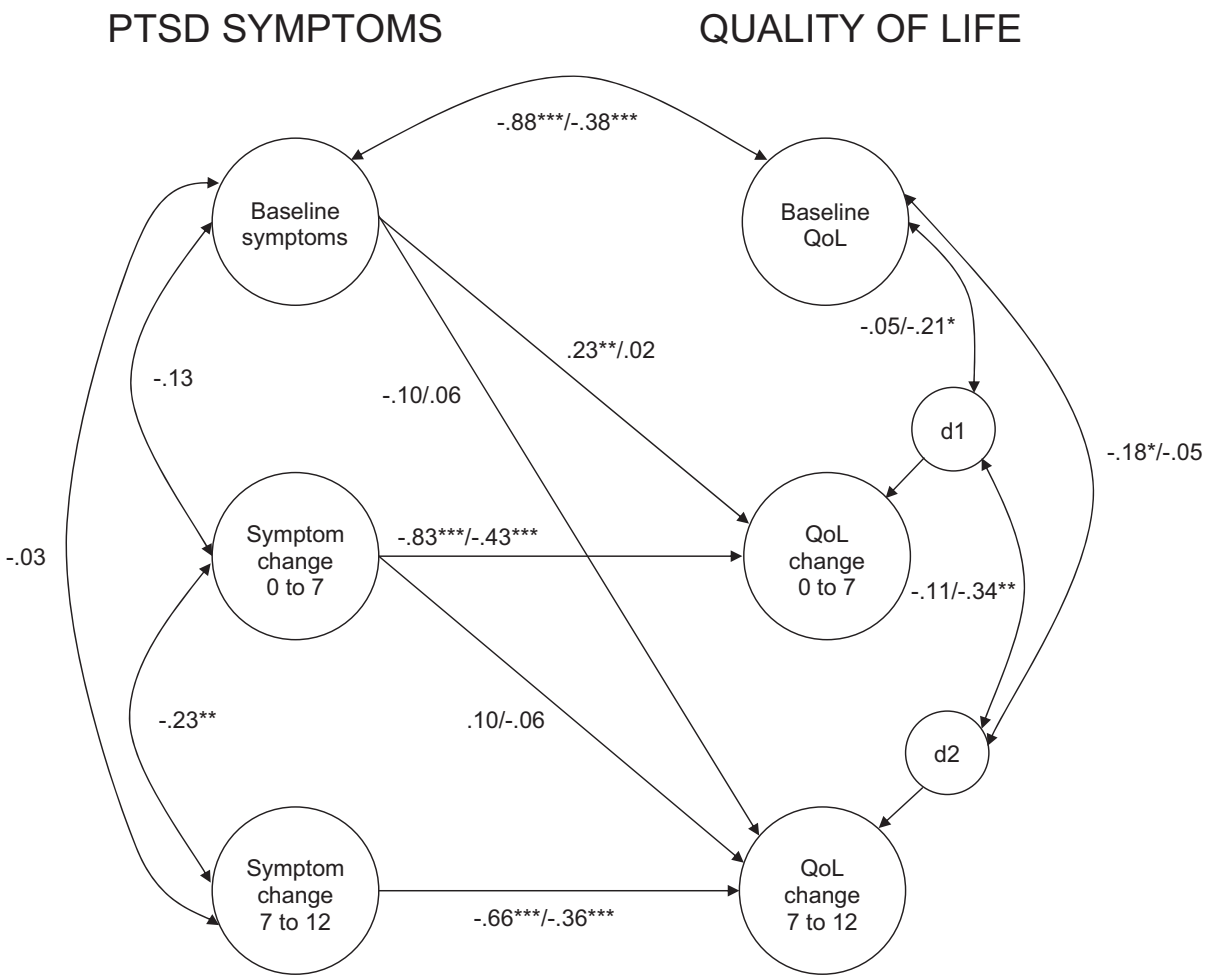

Figure 2. Structural model estimating change in psychosocial and physical health-related quality of life from baseline and change in posttraumatic stress disorder (PTSD) symptoms. Results for psychosocial quality of life (QoL) are presented to the left of the slash, with physical health-related QoL results presented to the right. Numbers beside bidirectional arrows are correlations, and unidirectional arrows are standardized path coefficients. $*p < .05$; $**p < .01$; $***p < .001$.

2). Higher PTSD symptom severity was associated with poorer quality of life at baseline. There were synchronous effects of PTSD symptom change during both treatment phases; symptom improvement was associated with improvement in quality of life. The lagged effect of symptom change on health-related quality of life was not statistically significant. Baseline PTSD symptoms did not predict change in the physical health domain during either phase. The magnitude of the relationship between PTSD symptoms and health-related quality of life was weaker than in the psychosocial domain. Overall, the model explained 19% of the variance in change in physical health–related quality of life from 0 to 7 months and 13% of the variance in change from 7 to 12 months.

In the analyses performed to examine causal paths from quality of life to symptoms, the reversed model for psychosocial quality of life fit slightly worse than the original model, $CFI = .96$, $RMSEA = 0.06$, $\chi^2(121) = 260.81$, $p < .001$. As in the original model, only the synchronous effects were significant. Baseline quality of life did not predict change in PTSD symptoms during either phase. For physical health-related quality of life, the fit of the reversed model was similar to the fit of the original model, $CFI = .98$, $RMSEA = 0.04$, $\chi^2(176) = 270.21$, $p < .001$. The only notable difference was a significant lagged effect from quality of life change from 0 to 7 months to PTSD symptom change from 7 to 12 months (standardized path coefficient $= -.20$, $p < .05$).

Discussion

To our knowledge, this is the first study to directly examine how change in PTSD symptoms relates to change in quality of life among individuals with PTSD. Higher PTSD severity was associated with poorer psychosocial and physical health–related quality of life at the beginning of treatment, a finding that is consistent with results of cross-sectional studies (e.g., Magruder et al., 2004; Rapaport et al., 2005). Although there were no overall improvements in quality of life among participants in the original treatment study (Schnurr et al., 2003), we found that there was a significant

amount of change within individual participants and that this change was related to PTSD symptom change. Our study builds on prior longitudinal investigations (e.g., Holbrook et al., 2001; Zatzick et al., 2002) by explicitly assessing how *change* in PTSD symptoms is related to *change* in quality of life. These relationships occurred synchronously, that is, during the same period of time. Initial symptom change was unrelated to later changes in quality of life.

More evidence is needed before firm conclusions about the absence of lagged effects can be drawn. Perhaps the minimal impact of treatment or the small average change in either symptoms or quality of life in the original study impaired our ability to detect such effects. However, we believe that this is an insufficient explanation given the marked intraindividual change and the substantial synchronous relationships we observed. Although lagged effects should be explored in a study with greater average treatment effects, it also would be useful to examine how PTSD symptom change relates to specific elements of quality of life (e.g., social, occupational), which might vary in response to the reduction in symptoms.

Results were consistent across psychosocial and physical domains of quality of life but were much stronger for the psychosocial domain. This may be due to the overall poor physical health of our sample, but additional factors may be relevant. It is easy to understand how reductions in nightmares, intrusive thoughts, and hypervigilance could affect life satisfaction or functioning. It is perhaps more difficult to understand how reducing PTSD symptoms could affect physical health, despite evidence that PTSD is associated with poor health (see Schnurr & Green, 2004). The discussion of mechanisms by which PTSD might affect health is beyond the scope of this article; biological, psychological, and behavioral factors are likely to be involved (Schnurr & Green, 2004). Nevertheless, our results extend current knowledge by showing how changes in PTSD symptoms are related to changes in physical health–related quality of life.

Our primary focus was unidirectional given the literature, which has emphasized the effect of PTSD symptoms on quality of life. In exploratory analyses to examine the effect of change in quality of life on symptom change, we observed findings similar to the primary results, although we also found a lagged effect of initial change in physical health–related quality of life on subsequent change in PTSD. It is likely that symptoms and quality of life interact mutually over time over time, with life events such as divorce and job loss worsening symptoms and symptom resolution leading to improved social and occupational functioning. An ideal context in which to address this issue would be a treatment study in which there were clinically meaningful treatment effects, objective and subjective quality of life measures, and more frequent measurement intervals to capture the dynamic interplay between symptoms and quality of life.

Caution in generalizing our findings to other PTSD samples is warranted. Our sample consisted of middle-aged male veterans with chronic PTSD. Two thirds were White, and 70% were receiving disability compensation. Prior to generalizing our findings to all PTSD patients, it would be useful to apply our methods to data from a more diverse and less impaired sample that showed greater average improvements in PTSD symptoms and quality of life.

Despite these limitations, our statistical approach is worth noting. The advantages of latent growth models to assess change as compared with difference score analysis are well known, the most important being more reliable measurement of change (e.g., Duncan, Duncan, Strycker, Li, & Alpert, 1999; MacCallum et al., 1997). Using multiple indicators of symptoms and quality of life in a latent variable measurement model allowed us to further improve reliability. Unlike studies that have examined whether treating PTSD symptoms improves quality of life, our approach allowed us to examine both synchronous and lagged effects and to quantify the magnitude of these effects in the context of optimal measurement reliability.

Our observation of the relationship between change in PTSD and change in quality of life complements evidence that successfully treating PTSD results in improved quality of life (e.g., Foa et al., 1999; Rapaport et al., 2002; Tucker et al., 2001). In their thoughtful discussion of quality of life and clinical significance, Gladis et al. (1999) provocatively asked, "Should clinicians and their patients feel that the job is not done (or not done well) if symptoms are alleviated but other areas of the patient's life are not fully satisfying?" (p. 328). The robust associations we observed between change in symptoms and change in quality of life suggest that interventions targeting quality of life would be unnecessary. However, it is important to remember that our data indicate relative relationships and not the absolute level of symptoms or quality of life attained. Also, we did not attempt to improve quality of life. Perhaps couples therapy or work therapy could lead to further improvements in these domains beyond that attained through resolution of PTSD symptoms. Future studies should examine whether treatments designed to enhance quality of life could confer additional benefit to patients beyond the benefit resulting from PTSD treatment.

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Received August 12, 2005
Revision received April 3, 2006
Accepted April 11, 2006 ■